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MINI-REVIEW

Surgical management of intractable lesional temporal lobe epilepsies

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Summary Structural lesions are found in about 30% of patients who undergo epilepsy surgery for intractable temporal lobe epilepsy. After detailed presurgical evaluations, the concordance of semeiology, focal structural lesions, ictal and interictal epileptiform discharges and neuropsychological dysfunction may offer favorable seizure control outcome postoperatively. The temporal lobe structural lesions are classified by magnetic resonance imaging-defined location into mesial temporal and lateral temporal (extrahippocampal) groups. In the lateral temporal group, subdural grid and depth electrode implantation is mandatory to localize the epileptic cortex and the possible secondary focus in the mesial temporal structures. Different surgical strategies, such as lesionectomy alone, lesionectomy with removal of adjacent epileptic cortex, lesionectomy with removal of mesiotemporal structures, have variable seizure control outcomes. The underlying pathology and the completeness of the lesion resection also affect the outcome.
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1. Introduction

The incidence of epilepsy ranges from 0.5% to 1.0% of the total population. Approximately one-third of epileptic patients have their seizures refractory to medical treatment. About half of the medically intractable focal epileptic patients are potential candidates for surgical treatment.[1] Epilepsy surgery for intractable temporal lobe epilepsy (TLE) with or without structural lesions usually provides favorable surgical outcome. Structural lesions are found in about 30% of surgical specimens resected for intractable TLE.[2,3] The proper evaluation of clinical semeiology, focal structural lesions, interictal and ictal electroencephalogram (EEG) abnormalities, and neuropsychological dysfunctions, especially when these factors are in concordance, may help to offer good seizure control postoperatively.[4] The presence of a structural lesion in a patient with intractable TLE does not always localize the epileptogenic zone. The temporal lobe lesion can induce

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secondary epileptogenicity in the mesiotemporal structures. The incidence of an extrahippocampal lesion and mesiotemporal sclerosis ranges from 8% to 22%.[5–8] Different surgical strategies, such as lesionectomy alone and lesionectomy with removal of mesiotemporal structures, have variable seizure control outcomes.[9–12]

2. Preoperative investigations

The preoperative investigations for intractable TLE consist of serial EEG recordings, long-term EEG/video monitoring with sphenoidal electrodes, magnetic resonance imaging (MRI), magnetic resonance spectroscopy (MRS), positron emission tomography with fluorodeoxyglucose (FDG-PET) and neuropsychological assessment.[13] MRI is the only investigation that can distinguish between substrate-related and substrate-unrelated epilepsy. An MRI-identified lesion is a strong predictor of favorable seizure outcome following surgery.[14] The sensitivity of MRI in detecting the structural lesions in refractory epileptic patients approaches 100%, with a reported specificity of 87%.[15] In our reported series,[13] MRI detected the lesion in all 12 patients in the vascular group, and in all 12 patients with neoplasms other than low grade astrocytomas in the neoplastic group. However, MRI detected the lesions in only 61% (14/23) of patients with low-grade astrocytomas. Won et al[16] reported that among MRI, PET and single photon emission computerized tomography in lateralizing epileptogenic foci, PET was the most sensitive tool which correctly lateralized the focus in 85% of the patients. PET may be used as a complementary tool in cases of inconclusive lateralization with other presurgical evaluations. Detection of the function of the mesiotemporal structure before operation is mandatory in the surgical strategy for lesional intractable TLE. High resolution, thin-cut (3 mm) slices MRI with a typical epilepsy protocol is recommended for the recognition of hippocampal sclerosis.[17] MRS can evaluate the biochemical and metabolic condition of the brain tissue. Reduction of the N-acetyl-aspartate (NAA) and decreased ratio of NAA/creatine plus choline complex indicates relative dysfunction of the measured area. MRS can provide evidence of hippocampal damage in a high percentage of patients with TLE. An extrahippocampal lesion in combination with MRS evidence of hippocampal sclerosis (dual pathology) can be found in up to 67% of patients with temporal neocortical epilepsy.[18]

3. Surgical strategies

The goal of epilepsy surgery in treating intractable lesional TLE is seizure freedom. It is necessary to remove the structural lesion as well as the epileptogenic cortex while preserving function. In Phase I preoperative investigations, the epileptogenic focus of epileptic activity may not correlate well with the abnormalities on MRI. Invasive Phase II studies using subdural grids, depth electrode implantation and intraoperative electrocorticography (ECoG) are mandatory to localize seizure onset.[19] A meta-analysis of simple excision versus epilepsy surgery was performed by Weber et al[20] in 1993. The authors concluded that in patients with lesional intractable seizures, epilepsy surgery had a better outcome of rendering the patient seizure free.

Table 1 Pathology in intractable TLE patients with structural lesions

Lesion type	No. of patients
Neoplastic group	82
Low-grade glioma	59
Astrocytoma	53
Oligodendroglioma	06
Anaplastic astrocytoma	08
DNET	03
Ganglioglioma	12
Vascular group	25
Arteriovenous malformation	11
Cavernous malformation	14

Lesional TLE/TLE = 107/420 = 25.5% (1987–2010 at Taipei Veterans General Hospital).
DNET = dysembryoplastic neuroectodermal tumor; TLE = temporal lobe epilepsy.

In general, without consideration of the location of the structural lesion, surgical strategies for intractable lesional TLE include: (1) lesionectomy alone; (2) lesionectomy with removal of the adjacent electrical abnormal cortex; (3) lesionectomy with removal of the mesial temporal structures (dual pathology); (4) removal of the electrical abnormal cortex alone; (5) anterior temporal lobectomy

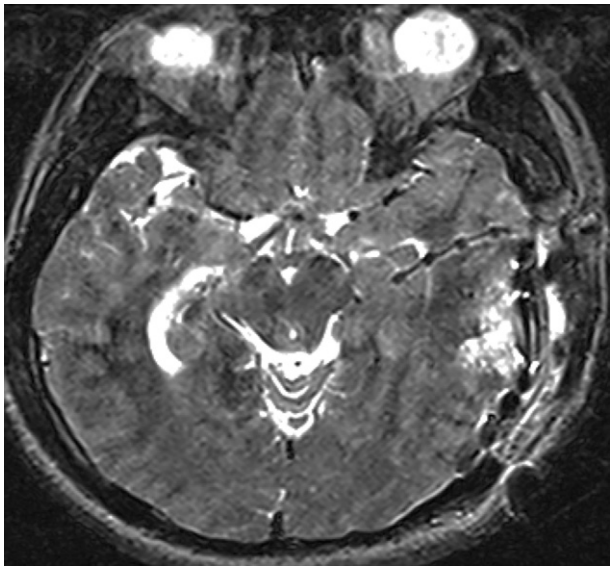

Figure 1 A 23-year-old male presented with intractable TLE for 3 years. An ill-defined 3.8 cm lesion, involving the gray and white matter of the left inferolateral midtemporal lobe, was noted with high signal on the FSE T2-weighted images axial MRI. Two subdural grids with 4 × 8 and 2 × 8 electrodes were implanted in the left temporal lobe and covered the entire lesion. A 4-contact depth electrode was implanted with the first contact precisely located at the left hippocampal head. FSE = fast spin echo; MRI = magnetic resonance imaging; TLE = temporal lobe epilepsy.

(ATL); and (6) disconnecting the pathway of seizure propagation. The epileptogenic cortex can be defined by epileptogenic discharges on intracranial ECoG or by the ictal onset zone localized by invasive intracranial EEG.[21] The completeness of the resection of the lesion, the underlying pathology and the extent of the removal of the associated epileptogenic cortex may affect the outcome of intractable lesional TLE.

Lesionectomy is defined as surgical removal of the structural lesion alone. However, pure lesionectomy is difficult in surgical removal of the discrete lesions such as cavernous malformations and arteriovenous malformations (AVMs). The adjacent gliotic epileptogenic brain tissues usually are removed during dissection and excision of the lesion. By contrast, the infiltrative lesions, such as astrocytomas and gangliogliomas and developmental cortical

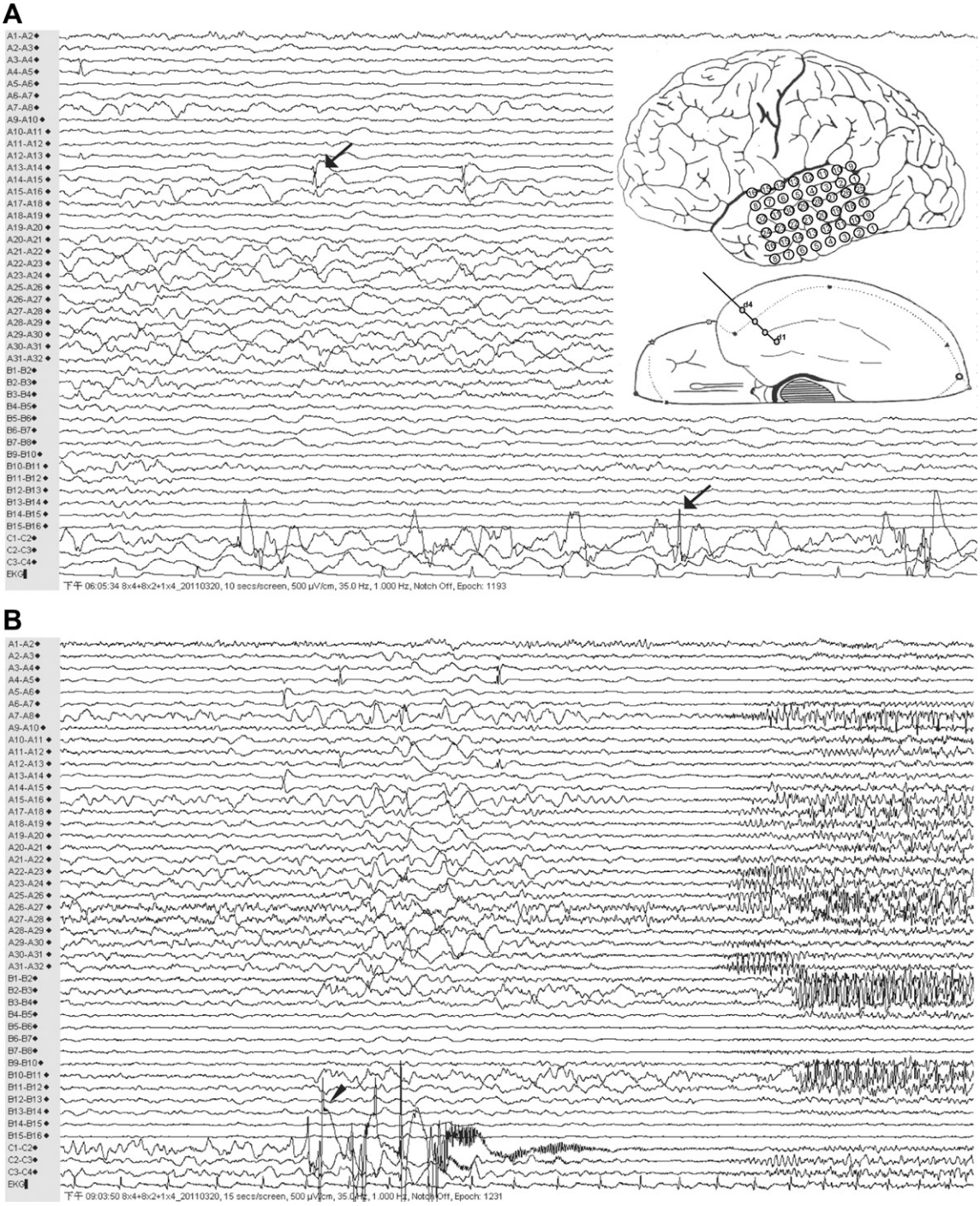

Figure 2 (A) The electrocorticogram revealed independent interictal spikes from the left lateral cortex around the lesion and the left hippocampus; (B) the ictal onset was initiated at the left hippocampus then spreading to the left lateral cortex surrounding the lesion.

anomalies such as cortical dysplasia, can be treated by pure lesionectomy without removal of adjacent normal brain tissue. When the lesion is inaccessible or located in an eloquent cortex, the alternative approach is to remove the epileptogenic cortex alone. Sometimes the lesion may be regarded as unrelated to seizure onset.

Temporal lobe structural lesions can be classified as: (1) developmental abnormalities including cortical dysplasia and heterotopias; (2) vascular lesions including AVMs and cavernous malformations; (3) tumors including astrocytomas, gangliogliomas and dysembryoplastic neuroectodermal tumor (DNET); and (4) encephalomalacia due to trauma or ischemia.[22] In our unpublished series, there were 107 (25.3%) cases of lesional TLE out of 420 cases of TLE operated upon in the period between 1987 and 2010 (Table 1). The structural lesions within the temporal lobes are classified by the location defined by MRI into the mesial temporal group and the lateral temporal (extrahippocampal) group. Different surgical strategies were employed to maximize preservation of functionally intact mesial structures in the treatment of epileptogenic mesial and lateral temporal lobe lesions.[23]

The mesial temporal group patient usually presents with mesial TLE (MTLE). MTLE is a well-defined clinical entity with a characteristic semeiology, an EEG pattern and pathological findings. Seizures usually consist of auras followed by oral and/or gestural automatisms, and sometimes secondary generalization. Some authors perform ATL, which may or may not imply complete lesionectomy and usually includes mesial structures. In most cases, ATL is associated with good seizure control outcomes, and up to 80% of patients turn seizure-free.[4,8–10,24] However, in terms of maximal resection of the structural lesion and seizure irritating zone while preserving hippocampal function, several alternative surgical procedures have been advocated.[23] If the structural lesion is separated from mesial structures on MRI and the hippocampus shows normal size and function, the lesion is resected while preserving mesial structures. In the nondominant hemisphere, mesial structures are removed despite a normal functioning hippocampus if the lesion is adjacent to or infiltrating the hippocampus. However, more conservative procedures should be considered in the dominant hemisphere.[23]

The lateral temporal (extrahippocampal) group patients with a structural lesion located within the neocortex varies in seizure characteristics. The neocortical temporal lobe epilepsies (NTLE) often present with olfactory and gustatory auras, complex gestures, ictal speech and secondary generalizations.[25] NTLE can be treated with a variety of surgical strategies, and may include resection of mesial temporal structures (dual pathology). Usui et al[26] reported the intracranial EEG findings in 15 patients with lesional lateral TLE. They routinely implanted subdural grids covering the lesion, and bilaterally placed depth electrodes in the hippocampi and amygdalas to confirm the origin of ictal discharges, and then decided upon the part of the brain that should be excised. They concluded that approximately 50% of the patients with structural lesions in the lateral cortex showed independent epileptogenic areas in ipsilateral mesial structures. Interictal spikes are not an indicator of whether mesial structures should be resected. In our institute, we implant a subdural grid covering the lesion and a depth electrode within the ipsilateral hippocampus (Figs. 1 and 2). The recordings of epileptiform discharges from the subdural grid and depth electrode can provide the information on ictal onset and the propagation pathway. Some authors have adopted surgical strategies using hippocampal atrophy on MRI as an indicator of whether to include the mesial temporal structures in surgical resection.[5,8,11] However, long-term extraoperative invasive intracranial EEG recording remains the most reliable method to confirm epileptogenicity.[21] After confirmation, surgical strategies fall into three major groups: (1) lesionectomy alone; (2) lesionectomy with removal of adjacent epileptogenic cortex; and (3) lesionectomy with removal of mesial temporal structures. If EEG recordings are concordant with the lesion, lesionectomy alone will result in a high rate of seizure freedom. Incomplete lesionectomy and resection of electrically active cortex alone seem less successful. The underlying pathology and extent of removal of the associated epileptogenic cortex also affect seizure control outcome.[20,22]

4. Conclusions

Seizure control outcomes for surgical treatment of lesional intractable TLE vary widely. Good results have been reported with lesionectomy alone (usually with removal of the adjacent gliotic cortex) in discrete lesions such as a cavernous malformation. The completeness of lesionectomy in infiltrative lesions such as astrocytoma, ganglioglioma and DNET and developmental cortical anomaly such as cortical dysplasia may alter the seizure control outcome postoperatively. In TLE patients who present with dual pathology, concomitant removal of mesial temporal structures helps to offer good surgical outcomes. With the advancement of diagnostic techniques, previously indolent lesions will become evident, and the seizure control outcome will improve because of clear delineation of lesions and the corresponding electrically abnormal cortex.

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